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Eyelid position and apposition

Bosch, Wilhelmus Antonius van den

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Summary

The position of the eyelids, and the cosmetic and functional relations between the eyelids and the eyeball are the subjects of this thesis. It discusses several causes of pathological eyelid laxity or abnormal eyelid position, which interfere with proper functioning of the eyeball, are cosmetically unpleasing, or both.

Section 1

The chapters in section 1 provide a survey of the anatomical and physiological backgrounds of the following sections of this thesis. The first chapter discusses the importance of data on the normal position of the eyelids and eyeball. Chapter two presents an overview of the anatomy of the upper eyelid. The causes and treatment of congenital and acquired ptosis are discussed. The next chapter provides additional information on the symptoms and treatment of Graves' orbitopathy. The possible influence of orbital decompression on the outcome of surgical correction of upper eyelid retraction is discussed. The final chapter presents current concepts about the relation between excessive upper eyelid laxity and chronic ocular discomfort, and about the causes and treatment of involutional entropion of the lower eyelid.

Section 2

Section 2 presents a study on topographic anatomy of the eyelids and the eyes, and of the influence of ageing on the position of these structures. Of 160 male and 160 female subjects aged between 10 and 89 years, slides were made of both eyes from anterior and lateral. These slides were used to measure the position of the eyes and eyelids.

The horizontal eyelid fissure lengthens more than 10% between the ages of 12 and 25. At middle-age, its length starts to decrease until, at the age of 85, the horizontal eyelid fissure again almost has the length it had at the age of 12. Ageing also causes a decrease of the distance between the lateral canthal angle and the anterior corneal surface, as viewed from

the side. Both changes are probably caused by laxity of the lateral canthal structures and/or atrophy of the orbital fat.

Ageing also causes a lower position of the lower eyelid relative to the pupil center, especially in men, and a higher position of the skin crease and of the eyebrows in both sexes. Ageing does not affect the position of the pupil center or of the lateral canthus.

Women show a slightly higher position of the upper eyelid skin fold, and a markedly higher position of the eyebrow, while the horizontal and vertical eyelid fissure are slightly larger in men. Many of these data were not yet available, especially regarding the influence of ageing on the position of eyelid structures.

Section 3

Section 3 contains three studies on the functional and cosmetic aspects of ptosis of the upper eyelid. Chapter 3.1 describes the results of the examination and treatment of 114 consecutive patients with congenital ptosis. Amblyopia was diagnosed in 20% of these patients, which is about five times more often than in the normal population. In only 20% of these cases, the amblyopia could directly be attributed to stimulus deprivation caused by the ptotic eyelid. In the remaining 80%, it occurred secondary to strabismus and refraction disorders. Of the whole ptosis group, 7% proved to be too old for successful treatment of their amblyopia at the moment of referral. With surgical treatment, a good result was obtained in 85% of the patients.

Because of the high incidence of amblyopia in patients with congenital ptosis, ophthalmological examination of these patients within the first year of life is indicated. To improve the knowledge about this disorder and thus to achieve quicker referral and treatment, these findings were published in the *'Nederlands Tijdschrift voor Geneeskunde'*.

Chapter 3.2 presents a study on the relation between the prolonged wearing of hard contact lenses and the occurrence of ptosis of the upper eye-

lid. On the oculoplastic department, we saw many patients younger than 50 years of age, who showed the clinical picture of aponeurogenic ptosis, while this disorder usually occurs at elderly age. All these patients had been wearing hard contact lenses for several years. To study the relation between prolonged hard contact lens wear and ptosis, at our contact lens department the position of the upper eyelids was measured in 50 consecutive subjects who had been wearing hard contact lenses for at least 10 years, and in 50 matched control subjects. About 10% of the contact lens wearers showed ptosis (defined as the average position of the upper eyelid in control subjects minus 2 sd).

Several mechanisms can tentatively be put forward to explain the relation between hard contact lens wear and ptosis, such as rubbing of the lens against the posterior side of the eyelid, or pulling on the eyelids in order to remove the lens from the eye. However, the precise cause remains a mystery.

Since the publication of our study, prolonged hard contact lens wear has become acknowledged to be the primary cause of acquired ptosis in the young and middle-aged population.

The mechanism that causes a high skin crease in aponeurogenic ptosis is the subject of chapter 3.3. The skin crease is the fold in the pretarsal upper eyelid skin that becomes visible when the eyes are closed. It is assumed to be created by fibres of the levator aponeurosis that insert subcutaneously. Patients with aponeurogenic ptosis show a higher upper eyelid skin crease than normal subjects. Its cause, however, was unknown.

Aponeurogenic ptosis is caused by disinsertion or thinning of the lowermost part of the levator aponeurosis. The orbital septum inserts on the anterior surface of the levator aponeurosis, just above the level at which the levator aponeurosis fibres insert subcutaneously. This insertion blocks the migration of orbital fat between the eyelid tissues. Consequently, the tissues of the anterior and posterior eyelid lamellae will always adhere below the level of insertion of the orbital septum. When the levator aponeurosis disinserts from the tarsal plate, the insertion of the orbital septum might migrate upward. Since the anterior and posterior lamellae of the eyelid will still adhere below this (higher) level, the skin crease would

become displaced upward. To study the existence of this causative mechanism, we measured the distance between the eyelid margin and the skin crease preoperatively in 25 consecutive patients suffering from aponeurogenic ptosis. During surgery, we measured the distance between the upper eyelid margin and the insertion of the orbital septum on the anterior surface of the levator aponeurosis. These results were compared to those of similar measurements in patients treated for their upper eyelid dermatochalasis.

Patients with aponeurogenic ptosis showed a higher skin crease, while both groups showed a significant relation between the height of the skin crease and the height of the orbital septum. This strongly suggests that the high skin crease in aponeurogenic ptosis is indeed caused by upward displacement of the orbital septum relative to the eyelid margin, as described above. This new anatomical concept explains problems with tissue identification during aponeurotic ptosis surgery.

Section 4

Section 4 deals with the relation between the position of the eyeball and cosmetic problems with the correction of upper eyelid retraction. This disorder mainly occurs in patients with Graves' orbitopathy. Next to hyperthyroidism, these patients suffer from various amounts of exophthalmos, strabismus, eyelid swelling, and upper and lower eyelid retraction. Treatment comprises several consecutive steps in a specific order. The thyroid dysfunction is treated first. Irradiation of the retrobulbar space can be performed to reduce the inflammation. The next step, if indicated, comprises treatment of the exophthalmos with orbital decompression. In this operation, the inferior and medial orbital walls are partially removed, allowing the orbital contents to prolapse into the adjacent paranasal sinusses. After treatment of diplopia, the final treatment step consists of surgical correction of any eyelid disorders, such as upper eyelid retraction.

With disinsertion of the upper eyelid retractors under local anaesthesia, the upper eyelid can usually be put in a normal position. This greatly improves the cosmesis, while ocular discomfort due to exposure keratitis also decreases.

In 14 out of 120 eyelids treated this way, the cosmetic result was unsatisfactory, although the position of the upper eyelid relative to the cornea was good. These eyelids showed a cosmetically unpleasing flat curvature, as viewed from anterior. This may be explained by assuming that downward displacement of the eyeball had occurred, either due to the disease or due to the orbital decompression.

The upper eyelid can be compared to the visor of a helmet, which rotates around fixed points. In the eyelid, these fixed points are situated at about the medial and lateral canthus. If the eyeball drops, the eyelid has to rotate further downward to cover an equal amount of cornea. Viewed from anterior, this will yield the impression of a flat upper eyelid curvature.

To study this alleged mechanism, we measured the distance between the pupil center and a reference line through the medial canthi in 120 eyes of decompressed and non-decompressed patients with Graves' orbitopathy.

These measurements were compared to similar ones obtained from 90 control eyelids.

On average, the distance between reference line and pupil center was 4.5 mm in normal eyes, and 2.3 mm after orbital decompression. The flat curvature of the upper eyelid occurred when this distance was less than 2 mm. This occurred only after orbital decompression.

Contrary to the currently held opinion that a large downward shift of the eyeball occurs as an infrequent complication of orbital decompression, our data show that orbital decompression induces a small downward shift of the eyeball in many patients. If, due to this, the distance between the pupil center and a reference line through both medial canthi becomes less than 2 mm, correction of upper eyelid retraction may cause a flat upper eyelid curvature. This can be prevented by not lowering the eyelid fully to its normal position.

Section 5

Section 5 describes eyelid disorders related to excessive upper and lower eyelid laxity. The clinical symptoms of the 'Floppy Eyelid Syndrome' (FES) illustrate that abnormal laxity of the upper eyelid may

cause corneal pathology, although the precise cause is, as yet, unknown. FES is an idiopathic and rare disease which occurs in middle-aged obese men. They suffer from chronic irritation of one or both eyes. On examination they show an easily evertible, rubbery lax upper eyelid and diffuse punctate keratitis. Typically, the symptoms disappear when the apposition between upper eyelid and eyeball has been restored by surgically shortening the upper and, if indicated, lower, eyelid.

In chapter 5.1, four patients are presented who suffered from symptoms similar to those described in FES, but otherwise did not fit within the classical description; these patients were neither middle-aged nor male nor obese. Nevertheless, upper eyelid shortening cured the symptoms in all cases. This strongly suggests that any disease that causes excessive laxity of the upper eyelid relative to the eyeball may cause symptoms similar to those of FES. Consequently, classical FES might be an easily identifiable subgroup within a larger group of patients in whom excessive upper eyelid laxity causes ocular symptoms. We propose to call the latter disorder 'LES' ('Lax Eyelid Syndrome'). Clinical experience learns that not every patient with lax eyelids develops LES. It is therefore likely that other disorders, such as tear film abnormalities, contribute to its occurrence.

To evaluate the causes of FES and LES, the eyelid tissue that was excised in 6 patients was examined histologically. The results are presented in chapter 5.2. In five of these six patients, the Meibomian glands of the tarsal plate proved infested with a mite, *Demodex brevis*. This mite causes atrophy of the Meibomian glands. On examination of 20 randomly selected eyelid blocks, excised in other patients for another reason, infestation with *Demodex brevis* was demonstrated in only 3 cases. The significantly higher incidence of this infestation in patients with FES or LES suggests that it might play a role in the pathogenesis of FES, by inducing changes of the rigidity of the tarsal plate and/or of the composition of the tear film. It cannot, however, be ruled out that the infestation occurs secondary to other pathological changes causing FES.

Chapter 5.3 presents a study on the influence of lower eyelid tightening on the results of surgical correction of involutional lower eyelid entropion.

SUMMARY EYELID APPPOSITION AND ROTATION

This disorder is caused by age-related laxity of several lower eyelid structures. Laxity of the capsulopalpebral ligament (the fibrous ligament that connects the eyelid tissues to the inferior rectus and oblique muscles) facilitates inward rotation of the eyelid margin. Laxity of pretarsal connective tissue may cause the orbicularis oculi muscle to slip into ('override') the pretarsal space. Due to this, the point of impact of the muscle shifts anteriorly in relation to the eyelid margin, hence its contraction will cause inward tilting of the eyelid margin. This will occur sooner when, due to ageing, the lower eyelid also shows horizontal laxity.

Surgical treatment may be performed using a well-established technique, described by Jones. In this procedure, the capsulopalpebral ligament is reinserted to the eyelid margin through an incision parallel to it. However, this procedure does not correct any co-existing horizontal lower eyelid laxity, while this might improve the results of surgery. To evaluate the influence of horizontal eyelid tightening, it was added to the original procedure in all patients who showed such laxity on clinical examination. Horizontal laxity proved to be present in a large majority of cases; it was diagnosed in 240 out of 266 treated eyelids. Compared to the small group of patients in whom this adjuvant correction had not been performed, the incidence of both transient and persistent overcorrection proved significantly lower. No influence on the recurrence rate was found, which may be due to the very low frequency of this complication.

Conclusion

The function of the eye, the position of the eyeball, the position and apposition of the eyelids, and facial cosmesis are tightly interrelated. Upper eyelid retraction and abnormal eyelid laxity interfere with the integrity of the corneal epithelium. Hard contact lenses, meant to correct visual acuity 'invisibly', may finally cause the reverse: ptosis that interferes with vision and is also cosmetically unpleasing. Patients with congenital ptosis frequently suffer from amblyopia. If not diagnosed and treated timely, this may cause irreversible poor vision. Orbital decompression usually improves facial cosmesis. Sometimes, however, it causes a downward displacement of the eyeball which contributes to a poor cosmetic result of the

correction of upper eyelid retraction. Cosmetic eyelid surgery may cause exposure keratitis that interferes with the function of the eye.

Because eyelid surgery often has both cosmetic and functional consequences, the eyelid surgeon should be able to evaluate the interaction between the function of the eye and the eyelids. Apart from this, the mastering of specific surgical techniques is necessary. Eyelid surgery should therefore be regarded as a subspecialty for which specific knowledge and technical skills are required.